

A Rare Case of Ileitis and Colitis

Raffaele Pezzilli*

Department of Gastroenterology, San Carlo Hospital, Potenza, Italy

ABSTRACT

A 77-year-old male patient experienced a seven Kg weight loss and 4-5 bowel movements per day with the greasy and foul-smelling stools. He had arterial hypertension treated with olmesartan 20 mg per day for the previous five years. A colonoscopy was normal and histology showed a collagenous colitis. For the failure of budesonide treatment he was admitted to our Department. An upper gastrointestinal endoscopy showed the disappearance of duodenal villi and histology showed severe atrophy of the duodenal villi with lymphocytic infiltrate in the epithelium and lamina propria.

A diagnosis of ileitis and collagenous colitis associated with malabsorption due to the olmesartan therapy was made. The long delay between the onset of Olmesartan therapy and the development of enteropathy suggests that the reaction is a localized, delayed hypersensitivity response which is cell-mediated and results in damage to the small intestinal brush border and collagenous colitis.

A 77-year-old male patient, non-smoker and consuming 50 gr of pure alcohol/day, experienced a seven Kg weight loss and 4-5 bowel movements per day with the greasy and foul-smelling stools. He had arterial hypertension treated with olmesartan 20 mg per day for the previous five years. Both coprocultures and examinations for parasites were negative; a colonoscopy was normal and histology showed a collagenous colitis. For the failure of budesonide treatment he was admitted to our Department. He was asthenic, apyretic and the abdomen was meteoric with normal peristalsis. Biochemical examinations revealed a white blood count of 11,240/mm³, hemoglobin 9.3 g/dL, INR 1.39, creatinine 1.31 mg/dL, sodium 127 mmol/L, potassium 2.1 mmol/L, calcium 7.7 mg/dL, albumin 3.1 mg/dL and C-reactive protein 8.73 mg/dL, amylase 311 IU/L (normal values <100) and lipase 75 IU/L (normal value <60), IgA levels were normal as well as the transglutaminase IgA. Fecal fat excretion was elevated (42 gr/24 hours) and fecal elastase-1 concentrations were normal. Computed tomography did not show any alteration of the pancreatic gland. An upper gastrointestinal endoscopy showed the disappearance of duodenal villi (Figure 1)

and histology showed severe atrophy of the duodenal villi with lymphocytic infiltrate in the epithelium and lamina propria (Figure 2).

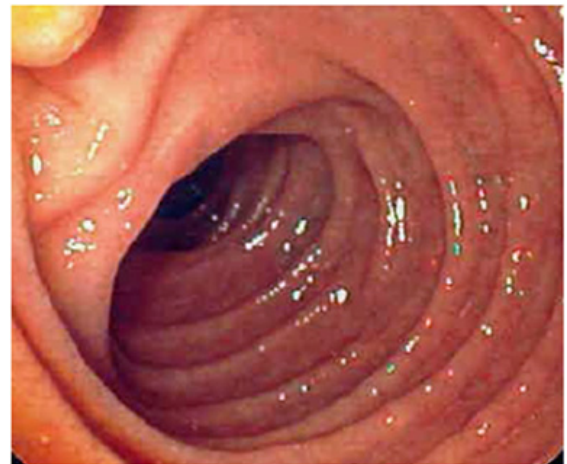


Figure 1: Upper gastrointestinal endoscopy showing the disappearance of duodenal villi.

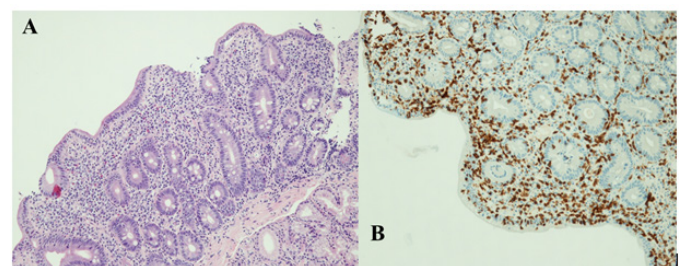


Figure 2: Severe atrophy of duodenal villi (HH 4x) (Panel A) and diffuse CD3 infiltrate of the epithelium and lamina propria (10x) (Panel B).

Correspondence to: Raffaele Pezzilli, Department of Gastroenterology, San Carlo Hospital, Potenza, Italy, Tel: +39-09711564229; E-mail: raffaele.pezzilli@gmail.com

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A diagnosis of ileitis and collagenous colitis associated with malabsorption due to the olmesartan therapy was made. The antihypertensive agent was discontinued and replaced with amlodipine 10 mg per day. The diarrhea progressively disappeared.

Olmesartan is an antihypertensive agent which belongs to the class of angiotensin II receptor blockers which has recently been described as a cause of drug-induced enteropathy resembling celiac disease (1). In addition, in some cases, an associated collagenous

colitis has also been reported (2). Symptoms include severe chronic diarrhea with substantial weight loss which may develop months to years after starting the drug. Physicians should be aware of this complication; a careful evaluation of the drugs taken by the patient admitted for chronic persistent diarrhea, significant weight loss with histological evidence of intestinal villous atrophy with serology negative for celiac disease and the presence of collagenous colitis should be carried out. The drug should immediately be discontinued. The long delay between the onset of Olmesartan therapy and the development of enteropathy suggests that the reaction is a localized, delayed hypersensitivity response which is cell-mediated and results in damage to the small intestinal brush border and collagenous colitis (2,3).

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